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By G. N. STEWART.

[From the Laboratory of Experimental Medicine, Western Reserve University.]

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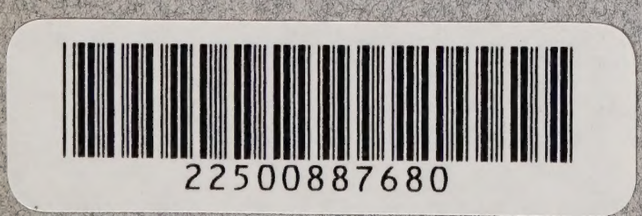

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THE INFLUENCE OF THE TEMPERATURE OF THE HEART ON THE ACTIVITY OF THE VAGUS IN THE TORTOISE.

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[*From the Laboratory of Experimental Medicine, Western Reserve University.*]

J. M. LUDWIG and B. Luchsinger[1] long ago concluded that in the tortoise, as in the frog, "not only does the vagus remain completely active at the highest temperatures which the heart can still endure, but it appears rather to have an increased activity at these lethal temperatures."

In an investigation of the influence of temperature and other factors on the heart and particularly on the action of the vagus and cardiac sympathetic nerves[2] in the frog, I incidentally made some observations on the inhibitory nerves of the ordinary European land tortoise, and concluded that "in general the inhibitory action of the right vagus of that animal is affected by the temperature of the heart in the same sense as that of the vagus in the frog; although the effect seems to be less marked than in the frog, and a much greater change of temperature is necessary to cause a sensible alteration in the inhibitory activity of the nerve. At very low temperatures it is unquestionably more difficult to obtain complete standstill of the heart than at the ordinary or at a higher temperature. But for a considerable range above and below the ordinary temperature it may be difficult to demonstrate any marked difference. It is by no means easy to show in the tortoise what is seen in the frog, that the minimum strength of stimulus needed to produce a given inhibitory effect increases as the temperature falls and decreases as the temperature rises. It needs a considerable fall of temperature to appreciably increase the minimum stimulus."

[1] Ludwig and Luchsinger: Archiv für die gesammte Physiologie, 1881, xxv, p. 211.

[2] Stewart: Journal of physiology, 1892, xiii, p. 59.

These conclusions have been called in question by E. G. Martin,[3] working with the terrapin, but confirmed by Bassin,[4] working in Kronecker's laboratory with the European land tortoise. Bassin, who does not seem to know of any work published in English, states that the strength of stimulation of the vagus which causes inhibition at the ordinary temperature causes as great an inhibition when the heart is heated to about 40°. Inspection of his curves and protocols shows indeed that complete inhibition may be obtained at the higher temperature with a strength of stimulation distinctly smaller than at the lower.

I have not been able to spare time to repeat the work, but, particularly since the appearance of Bassin's paper, hold this to be unnecessary. With the single exception of Lépine and Tridon,[5] all observers who have worked with European tortoises are agreed that the inhibitory activity of the vagus is, at any rate, as great at temperatures around 40° as at ordinary air temperatures. Why, then, has Martin reached the opposite result? (1) Chiefly, I think, because he has adopted a different criterion of vagus activity from the other observers cited. I am merely noting this difference, not criticising it. Ludwig and Luchsinger, Bassin and myself, determined a strength of stimulation sufficient to cause complete stoppage of the heart at the higher temperature, and then observed whether stoppage was caused by the same strength of stimulus at the lower temperature; or, *vice versa*, a strength of stimulation just insufficient to produce complete inhibition at the lower temperature was sought and its effect determined at the higher. The stimulation was kept up only long enough to produce a perfectly definite effect. Martin, on the other hand, in his first set of observations, "selected a strength of stimulus which would hold the heart at a practical standstill for several minutes at ordinary temperature, and then the effect of raising and lowering the temperatures was observed for this strength of stimulus." In the second set "advantage was taken of the fact that the heart of the terrapin can be maintained in standstill by stimulation of the vagus for a number of minutes without the effect of the stimulus becoming appreciably weaker. The heart was inhibited at ordinary tem-

[3] Martin: This journal, 1904, xi, p. 387.

[4] Bassin: Archiv für Anatomie und Physiologie, 1907, p. 429.

[5] Lépine and Tridon: Mémoires de la Société de Biologie, March 4, 1876.

perature, and then, while the stimulus was still on, the temperature of the organ was raised ten degrees or more." [6]

Now Martin found in general that at the higher temperature the heart during vagus stimulation gave a larger number of beats than at the lower temperature, or could not be so long prevented from beating. What was observed here was really the capacity of the heart to escape eventually from vagus control at the higher and lower temperatures, not the capacity of the vagus to cause complete, even if transient, inhibition, as in the experiments of the other writers.

The two tests could hardly be expected to give the same result. For if the vagus, acting upon an intracardial inhibitory mechanism whose excitability is increased by increase of temperature, is able to cause inhibition even more easily at high than at lower temperatures, it does not follow that the inhibition will be as long maintained. One would rather expect that the automatic "contractile energy," accumulating more rapidly at the high temperature in the automatic ganglia or the muscular fibres or in both, would sooner reach the threshold at which it overflows in spite of the continued stimulation of the inhibitory nerve. As I pointed out in my previous paper,[7] "although a high temperature is favorable to the initiation of inhibition, it is not necessarily favorable to its continuance. So far is this from being the case that the standstill obtained by chemical stimulation of the medulla oblongata at the ordinary temperature can be removed by gradually raising the temperature of the heart. And with electrical stimulation of the mixed vagus nerve the standstill, which is more easily obtained at a high temperature than at a low, often passes off sooner."

[6] Martin also made a few experiments in which he endeavored to see whether the minimal strength of stimulation needed to produce inhibition was altered by altering the temperature of the heart. The results, however, are such as are not easily interpreted. For example, in one experiment (he cites only two) he got no inhibitory effect at all with the strongest stimulation which could safely be employed when the temperature of the heart was 31.5°, although complete stoppage was caused with a much weaker stimulus at 22° before the heart was heated and at 23° after it was cooled again. If this is a typical result, the terrapin's heart must differ essentially in this respect from that of the tortoise, for I have never seen anything to suggest that a temperature of 31.5° will abolish the inhibitory power of the vagus, nor is there anything in the work of Ludwig and Luchsinger or of Bassin which even remotely indicates such a possibility.

[7] STEWART: *Op. cit.*, p. 99.

Even to this test, however, the vagus action at the higher temperature in Martin's experiments really was much more slightly impaired than would appear on a hasty comparison of the number of beats at the two temperatures during the period of vagus stimulation. For to get the true measure of the extent of the inhibition we must consider the number (and strength) of the beats the heated heart would have executed at the given temperature had the nerve not been stimulated. When, for example, we read that at 29° the heart beat 9 times in three minutes during stimulation of the vagus, whereas at 20° it contracted only thrice in five minutes, we must remember that, had it been left to itself, it would have beat probably not less than 90 or 100 times at the higher temperature and perhaps not half as frequently at the lower. The absolute amount of inhibitory effect, measured by the amount of work suppressed, is therefore very considerable at the higher temperature.

(2) Martin did not use such high temperatures (only up to 31° or 32°) as the other observers (up to 40° or a little more). Now, as already mentioned, I found that for a considerable range above and below the ordinary temperature it is not easy to demonstrate any marked difference in the minimal strength of stimulus which will cause inhibition. It is, of course, quite conceivable that the excitability of the intracardiac inhibitory mechanism or the capacity of the muscle fibres to respond to inhibitory stimuli may be a different function of the temperature at different temperatures in the same heart, and that the curves may not be the same for the hearts of different animals. Further, the relation between the effect produced on the contractile power of the heart and on the inhibitory mechanism by a given change of temperature need not be the same for all temperatures in one and the same or in different hearts. So that, if it is much easier, as I have shown, to demonstrate the progressive change in strength of the minimal stimulus required to produce inhibition for moderate increase or diminution of temperature from the ordinary temperature in the frog than in the land tortoise, there may also be a difference between the land tortoise of Europe and the terrapin in this regard.

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